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OFFERED REVIEW

Biological control of the soil-borne fungal pathogen *Sclerotinia sclerotiorum* –– a review

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Abstract
Diseases caused by *Sclerotinia sclerotiorum* (Lib) de Bary are difficult to control and cause increasing losses of horticultural crops worldwide. Reasons of this phenomenon are various: (i) the specialization of crop production that causes the accumulation of the pathogen in the soil; (ii) the lack of a safe and efficient method of soil fumigation; (iii) the specific life cycle of *S. sclerotiorum* with survival structures (sclerotia), resistant to chemical and biological degradation. Sclerotinia diseases depend on many environmental factors which determine sclerotia survival and ascospores dissemination, because plants are mainly infected by air-borne ascospores from carpogenic germination of sclerotia. Due to the lack of effective synthetic agents for eradication of *S. sclerortiorum* from soil considerable interest has been focused on biological control, especially the selection of microorganisms with mycoparasitic activity towards *S. sclerotiorum* sclerotia, that can decrease their number in the soil. In this work we review reports on the use of different antagonistic fungi and bacteria in the control of *S. sclerotiorum* and discuss the suppressive effect of organic amendments against this soil-borne pathogen.

Keywords Fungal pathogens · Sclerotia · Carpogenic germination · Antagonistic microorganisms

Introduction

Sclerotinia sclerotiorum (Lib.) de Bary [syn. *Whetzelina sclerotiorum* (Lib) Korf and Dumont 1972 (phylum Ascomycota)] is an ubiquitous pathogen of many plants belonging to the families Solanaceae, Cruciferae, Umbelliferae, Composite, Chenopodiaceae and Leguminosae (Kohn 1979; Willets and Wong 1980; Boland and Hall 1994), which was first reported from sunflower in 1861 (Purdy 1979). The fungus infects leaves, flowers, fruits and stems of the host plants, inducing diseases that can develop during the vegetation period or at the post-harvest stage, and cause severe losses to economically important crops in temperate regions of the world, mainly bean, carrot, pea, lettuce, mustard, canola, lentil and sunflower (Fernando et al. 2004; Clarkson et al. 2004; Del Rio et al. 2007). For example, in one major rapeseed cultivation area of China, *S. sclerotiorum* was reported to infect almost 4 to 7 million ha annually (Ni et al. 2014). The fungus overwinters in the soil or on crop debris as sclerotia, i.e. structures resistant to physical, chemical and biological degradation (Bolton et al. 2006).

Diseases caused by *S. sclerotiorum* are difficult to control because the long term persistence of sclerotia in the soil and the production of air-borne ascospores. Management of *S. sclerotiorum* occurs at several stages of crop development. Successful disease control usually requires implementation and integration of multiple methods. The environmentally least harmful are cultural practices that reduce the number of sclerotia in the soil. However, in many cases, especially for high value crops or highly specialized farms these methods are insufficient. Fungicides play the most important role in successful and effective white mold management (Mueller et al. 2002a; Vieira et al. 2003; Paula Junior et al. 2009; Derbyshire and Denton-Giles 2016). For instance, soil fumigation with metham-sodium decreased the amount of resting propagules (Ben-Yephet et al. 1986). Fungicides applied during the bloom period are effective in inhibiting infection by ascospores in fields with a history of diseases caused by *S. sclerotiorum.* Several chemical agents registered in the USA, Canada, Australia, Europe and China are available to this purpose. Their active ingredients are: boscalid, fluazinam, fluxapyroxad, pyraclostrobin, penthiopyrad, picoxystrobin,

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prothioconazole, prothioconazole and trifloxystrobin, tetraconazole, thiophanate methyl (Matheron and Porchas 2004; Bradley et al. 2006; Zhou et al. 2014a, 2014b; Wang et al. 2015; Derbyshire and Denton-Giles 2016). The effective control of *S. sclerotiorum* requires application of fungicides during sensitive time frames, the number of treatments depending on the length of the crop vegetation period and on how long flowers or petals are available for infection by ascospores (Heffer Link and Johnson 2007). More applications are needed for plants with longer bloom periods. Although some foliar-applied herbicides containing lactofen as the active ingredient have efficacy against *S. sclerotiorum*, their use may result in crop damage and yield reduction (Heffer Link and Johnson 2007). The use of fungicides to control sclerotinia stem rot of oilseed rape was reviewed by Derbyshire and Denton-Giles (2016).

The best way to limit pesticide application would be the use of cultivars resistant to *S. sclerotiorum*. However, due to the specific character of the diseases caused by this pathogen, breeding programs had a limited success so far (Yanar and Miller 2003; Otto-Hanson et al. 2011; Barbetti et al. 2014; Uloth et al. 2014). One promising way to obtain *S. sclerotiorum* resistant plants is the implementation of genetic engineering strategies by the use of host-induced gene silencing methods (HIGS) (Andrade et al. 2016). Studies with transgenic plants with increased level of resistance to *S. sclerotiorum* have also been conducted (Liu et al. 2015).

In the absence of resistant cultivars and environmentally friendly methods for the eradication of *S. sclerotiorum* from soils, research on biological methods was initiated, among which the application of antagonistic microorganisms and organic amendments, as discussed in the present review.

Pathogen biology

Sclerotia formation *S. sclerotiorum* is capable of reproducing both asexually (myceliogenic germination of sclerotia) and sexually (carpogenic germination of sclerotia) (Aldrich-Wolfe et al. 2015). On diseased plants, the fungus forms a white fluffy mycelium (white mold) and, after several days, it produces survival structures: the sclerotia (Ordonez-Valencia et al. 2014). These are black, melanized structures of different size which, depending on the host, range from a few millimeters (bean) to a few centimeters (sunflower) in length (Bolton et al. 2006). Sclerotia can germinate myceliogenically or carpogenically (Williams and Stelfox 1980); in the first case forming hyphae, in the second producing apothecia and, subsequently, ascospores.

Sclerotia are built from two (Willets and Wong 1971) or three (Arseniuk and Macewicz 1994) layers: rind, cortex and medulla each made up of a thick layers of hyphal aggregates. The outer ring is composed of cells whose walls contain the black compound melanin (Butler et al. 2009). This is a macromolecule composed of various types of phenolic or indolic monomers that protects fungi from harsh environmental conditions, i.e visible or ultraviolet light, toxic metals or lytic enzymes, and antagonistic microorganisms (Butler and Day 1998). *S. sclerotiorum* melanin is extraordinarily resistant to chemical degradation. The inner part of the sclerotium, the medulla, is embedded in a fibrillar matrix composed of carbohydrates and proteins (Le Tourneau 1979).

The morphogenesis of *S. sclerotiorum* sclerotia was described by Ordonez-Valencia et al. (2014). The first step of sclerotial formation (sclerotial primordial) was observed by these authors after four days of fungal growth in Petri dishes, when mycelium completely covers the surface of the medium. The aggregation of aerial hyphae was observed at the edge of plates, probably as a response to the limited nutrient availability. Next, during the development stage, the hyphae coalesced and became compacted. During the maturation period, the surface of the sclerotia became pigmented, due to melanin production in the ring cells, and acquired a rough texture. The described steps of sclerotia formation were observed on agar medium in laboratory conditions. It is possible that similar processes take place on diseased plants. However, it cannot be excluded that some differences may occur, depending on the environmental conditions and plant species.

The most important factor stimulating sclerotia formation is nutrient limitation. It was found that the vegetative growth of *S. sclerotiorum* was prolonged and sclerotium formation delayed in a medium continually supplemented to maintain a high energy status (Christias and Lockwood 1973) and it was also observed that under nutrient deprivation, carbohydrates and nitrogen were translocated to sites of sclerotial synthesis (Cooke 1971). Townsend (1957) demonstrated that the time of maturation of *Sclerotium rolfsii* sclerotia was related to the time of depletion of carbohydrates in the growing media. Rollins and Dickman (2001) showed that under neutral or alkaline pH sclerotial formation is inhibited. In an earlier work they described the role of 3',5'-cyclic monophosphate (cAMP) in the start of the phase from mycelial growth to sclerotia formation (Rollins and Dickman 1998). The molecular basis of sclerotiogenesis was described by Bolton et al. (2006).

Sclerotia survival Sclerotia have the capability of remaining viable for long periods of time because they are resistant to chemical and physically adverse conditions, as well as to biological degradation (Merriman 1976; Wu et al. 2008). Cook et al. (1975) showed that 78% of the sclerotia survive for at least three years when buried in uncultivated soil, while Cosic et al. (2012) demonstrated that after three years, the percentage of viable sclerotia can be up to 100%. There are reports that sclerotia can survive for up to 4-5 years (Adams and Ayers 1979) and many studies show the effect of burial depth, moisture and temperature on survival of *S. sclerotiorum*

sclerotia in the soil (Moore 1949; Merriman et al. 1979; Matheron and Porchas 2005; Wu et al. 2008; Duncan et al. 2006). Among these factors, the most detrimental one seems to be flooding. Under this condition, sclerotia may decay completely within 24-45 days (Moore 1949) or 14-21 days (Matheron and Porchas 2005). Cosic et al. (2012) showed that in the case of continuous flooding sclerotia buried in the soil at 5 cm depth were completely destroyed.

Sclerotia survival is strongly dependent on the depth at which they are buried in the soil. Information on the survival of sclerotia within the vertical soil profile is contradictory. Cosic et al. (2012) showed that in undisturbed soil sclerotia placed deeper (10-30 cm) stay alive longer than those in upper soil (5 cm). Similar results were obtained by Cook et al. (1975), who concluded that sclerotia from upper layers were degraded faster than sclerotia placed deeper in the soil profile, whereas Duncan et al. (2006) showed that the viability of *S. sclerotiorum* sclerotia, buried at 0, 5 and 10 cm, decreased with depth. This phenomenon occurred regardless of the timing of sampling in the growing season.

Carpogenic germination - ascospore formation As mentioned above sclerotia can germinate myceliogenically or carpogenically (Williams and Stelfox 1980). In the first case, the hyphae produced by sclerotia can directly infect plants. In the second case, sclerotia produce apothecia and subsequently ascospores. From each sclerotium, one or several apothecia can emerge. An apothecium is a structure consisting of a stipe topped with a discoid receptacle that bears a flat to concave hymenial layer with rows of asci. Each ascus contains eight hyaline, ellipsoid and binucleate ascospores (Kohn 1979). Apothecia develop rapidly on sclerotia located at the surface or near the surface of the soil.

Factors affecting carpogenic germination of *S. sclerotiorum* have been described in many papers (Schwartz and Steadman 1978; Caesar and Pearson 1983; Dillard et al. 1995; Sun and Yang 2000; Matheron and Porchas 2005; Wu and Subbarao 2008). The most important are moisture and temperature. Favorable temperature conditions mostly range from 10 to 20°C. However, temperature requirements are dependent on the origin of the *S. sclerotiorum* isolates and the temperature at which the sclerotia are produced (Huang and Kozub 1991). Sclerotia should be conditioned at low temperatures for some time to overcome dormancy and germinate carpogenically (Dillard et al. 1995). Those present in a dry environment are unable to germinate carpogenically. Wu and Subbarao (2008) observed that in a greenhouse, a 10- to 20-day dry period completely inhibited carpogenic germination, whereas maximum carpogenic germination was observed in fully water-saturated sclerotia (Nepal and del Rio Mendoza 2012). Sclerotia buried in soil were fully water-saturated at different times, depending on their size. Small, medium and large sclerotia were fully saturated within 5, 15 and 25 h, respectively (Nepal and del Rio Mendoza 2012). No apothecia formation was observed below 70 to 80% saturation. Clarkson et al. (2004) showed that carpogenic germination of sclerotia occurred between 5 and 25°C, but only when the soil water potential was >-100kPa. Above 26°C no apothecia were produced. In formed apothecia, the maturation of asci takes about 72-84 h (Clarkson et al. 2004). Ascospore maturation is a complex process that is influenced by multiple factors. Of all the factors studied, the temperature influences the process of ascus maturation significantly, the optimum temperature being 21°C. Apothecia discharge ascospores (about $2x10^6$ per apothecium) during a sudden decrease of atmospheric humidity or pressure (Wu et al. 2007). The hyaline, unicellular ascospores have thin walls and may survive for only a few days.

Disease development

Symptoms caused by *S. sclerotiorum* differ among host species (Lumsden 1979; Morrall and Dueck 1982; Steadman 1983; Patterson and Grogan 1985; Kora et al. 2003; McLaren et al. 2004). However, the most common symptoms, as for example in lettuce or beans, are water-soaked irregular spots and a characteristic white cotton-like mycelium present on leaves, stems, fruits and petioles. Next appear secondary symptoms such as water soaked lesions, wilting, bleaching and shredding of plants. At later stages of disease development, sclerotia are formed on the outer surface of affected plant tissues which, together with the decomposed plants, are transferred into the soil. Sclerotia may germinate and directly produce mycelium. The mycelium of *S. sclerotiorum* may directly infect plants growing close to the sclerotium. Diseases initiated by mycelium were observed in some vegetables such as carrots, lettuce, beans or sunflower (Steadman 1983; Hunter et al. 1984; Nelson et al. 1989; Kora et al. 2003). In sunflower, myceliogenic germination can cause a serious disease called sunflower wilt. In this case, infection occurs through the roots and progresses up into the stem (Nelson et al. 1989). Mueller *et al.* (2002b) demonstrated that the density of sclerotia in soil and the severity of infection decreased after deep plowing.

It is very difficult to assess the number of sclerotia that may be dangerous for any given crop. According to the Suzui and Kobayashi (1972), 3.2 sclerotia per m^2 may cause 95% infection of kidney bean in the field whereas Schwartz and Steadman (1978) showed that the minimum number of sclerotia to incite a moderately severe disease of dry edible bean (*Phaseolus vulgaris*) is 0.2 sclerotia/kg of soil.

More common and dangerous for plants is the carpogenic germination of *S. sclerotiorum*. Ascospores released by apothecia can be disseminated by air currents over several kilometers (Sedun and Brown 1987). Plants are primarily infected by air-borne ascospores from carpogenic germination

of sclerotia. For these reasons disease incidence is often sporadic and dependent on weather conditions favouring the production of apothecia (Hudyncia et al. 2000).

S. sclerotiorum infection and mycelium development is maximized in the presence of free water on the plant surface. Ascospores can germinate on the surface of healthy tissue but cannot infect plants without an exogenous nutrient source (Bolton et al. 2006),which often is provided by senescing leaves and petioles or juices flowing from the damaged plants (Kora et al. 2003). Thus, flowering is a particularly dangerous moment because senescing flowers serve as nutrient source for the pathogen (Turkington and Morrall 1993; Almquist and Wallenhammar 2015). Direct penetration of fungal hyphae was observed through the cuticle within 12 h from inoculation. The host cells were completely colonized by fungal mycelium 48 h after inoculation leading to tissue collapse (Davar et al. 2012). Upon establishment on the surface of plants, the fungus secretes pathogenicity factors: cell-wall and plant tissues degrading enzymes such as pectinases, cellulases, beta-1,3-glucanases, xylanases and glycosidases (Cotton et al. 2003; Bolton et al. 2006). These enzymes facilitate penetration of the fungus inside the plant and maceration of tissues. In the first step the main role is played by pectinases, because pectins are the main component of cell walls. *S. sclerotiorum* produces several forms of pectinases. Expression of genes encoding the fungal lytic system is regulated by ambient pH. Marciano et al. (1983) showed that an optimum production of pectinolytic enzymes occured at pH 4-5.

Infection of plants by *S. sclerotiorum* causes also the secretion of oxalic acid into plant tissues and pH reduction (Guimaraes and Stotz 2004). Cotton et al. (2003) demonstrated that the secretion of polygalacturonases and a decrease of pH are the results of oxalic acid production. Oxalic acid is important for pathogenesis of *S. sclerotiorum* and sclerotia formation (Cessna et al. 2000; Williams et al. 2011). Godoy et al. (1990a) showed that a *S. sclerotiorum* mutant unable to produce oxalic acid was also unable to produce sclerotia and was non-pathogenic to plants. Oxalic acid can suppress the oxidative burst of infected plants (Cessna et al. 2000), can induce apoptotic-like programmed cell death (Williams et al. 2011) and is decomposed by oxalate oxidase, an enzyme that has multiple impacts on plant cells (Wang et al. 2015).

Biological control

The activity of the biocontrol agents in the soil is affected by many abiotic and biotic environmental factors, e.g. temperature, water potential, pH, pesticides, organic matter, soil microorganisms, plant species and so on, making these agents usually less effective than synthetic pesticides. However, due to the less harmful effect on the environment and the lack of the effective chemical methods, safer biological methods are being sought.

Antagonistic microorganisms

Fungi *S. sclerotiorum* sclerotia are the most important survival structures of the pathogen in the soil. So, considerable interest has been focused on the selection of microorganisms which can neutralize these structures in the soil (Jones and Watson 1969). Many fungi showed mycoparasitic activities towards *S. sclerotiorum*. The results of these studies were described in many papers and are summarized in Table 1. Particularly intense studies were conducted with the parasitic fungus *Coniothyrium minitans* (Huang and Hoes 1976; Turner and Tribe 1976; McQuilken et al. 1995; Zeng et al. 2012b). Contans®WG, a commercial formulation of *C. minitans (strain CON/M/91-08)*, is known for its capacity to reduce the damage caused by *S. sclerotiorum* to several crops by infecting and degrading sclerotia in the soil (McQuilken and Chalton 2009). Target plants for treatment with *C. minitans* are high value crops as peanuts, sunflowers, lettuce, cucumber, beans and oilseed rape (EFSA 2016). Li et al. (2005) showed that three applications of *C. minitans* conidia ($5x10^6$ ml^{-1}) to alfalfa blossoms effectively suppressed sclerotinia pod rot in field conditions. The percentage of diseased pods in the *S. sclerotiorum*- infested treatment was 64, 42 and 72% and 38, 30 and 29% in the *C. minitans* treatment during three consecutive years. By spraying a *C. minitans* spore suspension on bean plants during blooming, the incidence of white mold was reduced by 56% (Huang et al. 2000). Also, incorporation of *C. minitans* in the top soil before planting of soybean reduced the disease severity index (DSI) by 68% and the number of sclerotia in the soil by 95.3% (Zeng et al. 2012a).

C. minitans produces a broad range of cell wall-degrading enzymes such as chitinases and glucanases as well as secondary metabolites like macrosphelide A, benzofuranones and chromanes (Tomprefa et al. 2011), that enhance colonization and degradation of *S. sclerotiorum* sclerotia. Direct penetration of sclerotia, degradation and disintegration of sclerotial tissues by *C. minitans* was also demonstrated (Tu 1984; Bitsadze et al. 2015). This mycoparasitic activity is affected by factors such as temperature and pH. Colonization of sclerotia by *C. minitans* occurred very fast and half of the sclerotia were infected during the first week. After four weeks 100% of the sclerotia were colonized (Zeng et al. 2012b). The optimum parameters for *C. minitans* growth were 15-20°C and pH 4.5-5.6.

Fungi of the genus *Trichoderma* are used extensively as biological control agents (BCAs) (Benitez et al. 2004; Harman et al. 2004; Vinale et al. 2008; Druzhinina et al. 2011; Hermosa et al. 2012; Aleandri et al. 2015). Many experiments conducted all over the world demonstrated parasitism of *S. sclerotiorum* sclerotia and reduction of apothecia

Table 1 Fungi showing mycoparasitic and antagonistic activity towards *S. sclerotiorum*

Species	References
Alternaria alternata	Inglis and Boland 1992
Aspergillus niger	Rai and Saxena 1975
Aspergillus ustus	Rai and Saxena 1975
Coniothyrium minitans	Tribe 1957; Huang and Hoes 1976; Turner and Tribe 1976; Whipps and Budge 1990; Budge and Whipps 1991; Trutmann et al. 1980; Tu 1984; Huang et al. 2000; Jones and Whipps 2002; McQuilken et al. 2003, McQuilken and Chalton 2009; Gerlagh et al. 2003; Chitrampalam et al. 2008; Jones et al. 2011; Zeng et al. 2012b; Bitsadze et al. 2015; Jones et al. 2015
Drechslera sp.	Inglis and Boland 1992
Epicoccum purpurascens	Zhou and Reeleder 1989; Inglis and Boland 1992
Fusarium graminearum	Inglis and Boland 1992
Fusarium heterosporum	Inglis and Boland 1992
Fusarium oxysporum	Rodriguez et al. 2006
Gliocladium virens	Tu 1980; Phillips 1986; Whipps and Budge 1990; Budge et al. 1995
Gliocladium roseum	McCredie and Sivasithamparam 1985
Microsphaeropsis ochracea	Bitsadze et al. 2015
Myrothecium verrucaria	Inglis and Boland 1992
Penicillium citrinum	Rai and Saxena 1975
Penicillium funiculosum	Rai and Saxena 1975
Penicillium pallidum	Rai and Saxena 1975
Sporidesmium sclerotivorum	Ayers and Adams 1979; Adams and Ayers 1983; Adams and Fravel 1990; Fravel 1997; Del Rio et al. 2002
Streptomyces lydicus	Zeng et al. 2012b
Talaromyces flavus	McLaren et al. 1983
Teratosperma oligocladum	Adams and Ayers 1983
Trichoderma asperellum	Geraldine et al. 2013; Aleandri et al. 2015
Trichoderma hamatum	Aleandri et al. 2015; Jones et al. 2015
Trichoderma harzianum	Bin et al. 1991; Budge and Whipps 1991; Knudsen et al. 1991; Menendez and Godeas 1998; Elad 2000; Chitrampalam et al. 2008; Zeng et al. 2012b; Steindorff et al. 2014; Aleandri et al. 2015
Trichoderma atroviride	Li et al. 2005; Matroudi et al. 2009
Trichoderma koningii	Castro 1995
Trichoderma virens	Zaidi and Singh 2013; Aleandri et al. 2015; Jones et al. 2015
Trichoderma stromatica	Paula Junior et al. 2009
Ulocladium atrum	Li et al. 2003

density by *Trichoderma* isolates (Geraldine et al. 2013). Most of these experiments were conducted under laboratory or greenhouse conditions (Matroudi et al. 2009; Smolinska et al. 2016). However, the number of reports dealing with the antagonistic activity of *Trichoderma* in field conditions is rather limited (Knudsen et al. 1991; Zeng et al. 2012a; Geraldine et al. 2013).

Geraldine et al. (2013) observed a reduction of *S. sclerotiorum* apothecia number and disease severity after application of *T. asperellum* at the dose of 2×10^{12} spores ml^{-1} per plot in two years of field experiments with common bean. A positive effect was also observed of *Trichoderma* treatment on the number of pods per plant and an increase of yields up to 40% compared to the control. *T. hamatum* reduced Sclerotinia disease of cabbage by 31-57% in field experiments conducted by Jones et al. (2015) showing that *T. hamatum*-colonized sclerotia had reduced apothecial production and a lower carpogenic infection of cabbage. The white mould of cucumber fruit and stems was reduced by 64 and 30-35%, respectively, after *T. harzianum* T39 application under commercial greenhouse conditions (Elad 2000). Another isolate of *T. harzianum* T-22, protected soybean against *S. sclerotiorum* and decreased the disease severity index (DSI) by 38.5% in a field-grown crop (Zeng et al. 2012a).

The mechanisms involved in the control of pathogenic fungi by *Trichoderma* include mycoparasitism (Zeilinger and Omann 2007; Geraldine et al. 2013), antibiosis (Elad 2000; Vinale et al. 2008) and systemically induced resistance (Harman et al. 2004; Nawrocka and Małolepsza 2013). Also, several studies have shown that isolates of *Trichoderma* spp. can significantly stimulate the growth of different plant species (Vinale et al. 2008; Smolińska et al. 2014). Fungi of the genus *Trichoderma* are characterized by rapid growth and abundant production of spores, so they are highly competitive compared with other soil-borne microorganisms. The ability to secrete active compounds varies greatly among *Trichoderma* species, and isolates. Mechanisms used by *Trichoderma* spp. in biological control vary with the species, pathogen and host plant. In the case of antifungal activity against *S. sclerotiorum*, the mycoparasitic properties of *Trichoderma* play an important role. Chitynases, glucanases, proteases and cellulases were identified among the *Trichoderma* enzymes that disintegrate the cell wall of the pathogens (Chet et al. 1998; Kaur et al. 2005; Zeilinger and Omann 2007; Lopez-Mondejar et al. 2011).

Antagonistic microorganisms

Bacteria Various studies have reported the capacity of diverse bacterial genera, such as *Bacillus* and *Pseudomonas,* to control fungal diseases (Table 2). Studies of biological control in the phyllosphere of host plants with bacterial antagonist were conducted much less frequently than with fungi (Fernando et al. 2007; Saharan and Mehta 2008). It was observed that *Pseudomonas chlororaphis* and *Bacillus amyloliquefaciens* significantly reduced stem rot of canola caused by *S. sclerotiorum* under field conditions (Fernando et al. 2007). The percentage of stem rot incidence after application of bacteria ($9x10^8$ CFU ml^{-1}) at 30-50% bloom stage, was 7.5-28.7% for *P. chlororaphis* and 5.0-29.6% for *B. amyloliquefaciens* in two field trials, and were significantly different from that of the pathogen-inoculated control (20.0-75.0%). Application of *P. chlororaphis* at 10^4 -10^8 CFU ml^{-1} inhibited ascospore germination of *S. sclerotiorum* on canola petals. When bacteria were applied prior to, or at the same time as *S. sclerotiorum*, there was a complete inhibition of the disease (Savchuk and Fernando 2004).

The antifungal activity of *Pseudomonas brassicacearum* DF41 (Loewen et al. 2014) against *S. sclerotiorum* under greenhouse and field conditions was demonstrated by Savchuk and Fernando (2004) and Berry et al. (2010). In later papers Berry *et al.* (2012, 2014) described the role of lipopeptide sclerosin produced by *P. brassicacearum* DF41 in the suppression of *S. sclerotiorum* and studied the role of quorum sensing and biofilm formation on production of antifungal compounds.

Antagonistic bacteria inhibit the germination of ascospores either through the production of antimicrobial substances or direct growth on ascospores. Fernando et al. (2007) suggested that application of *P. chlororaphis* on host plants induced systemic resistance against *S. sclerotiorum*. Strains of *Pseudomonas* spp. produce many antimicrobial compounds, i.e. pyoluteorin, pyrrolnitrin, phenazines, siderophores, cyanide, 2,4-diacetylphloroglucinol (Compant et al. 2005) and enzymes that can lyse fungal cells, i.e. cellulose, chitinase, proteases and beta-glucanase (Hernandez-Leon et al. 2015). In most cases *Pseudomonas* showed multiple mechanisms in the biocontrol of diseases caused by *S. sclerotiorum*.

Table 2 Bacteria with antagonistic activity towards *S. sclerotiorum*

Species	References
Bacillus subtilis	Zazzerini 1987; Zhang and Fernando 2004a; Chitrampalam et al. 2008; Hu et al. 2011; Zeng et al. 2012a, b; Monteiro et al. 2013; Hu et al. 2014; Kamal et al. 2015
Bacillus megaterium	Hu et al. 2013; Hu et al. 2014
Bacillus amyloliquefaciens	Fernando et al. 2007
Bacillus cereus	Zazzerini 1987; Kamal et al. 2015
Erwinia herbicola (Pantoea agglomerans)	Godoy et al. 1990b; Yuen et al. 1994
Pseudomonas chlororaphis	Zhang and Fernando 2004b; Savchuk and Fernando 2004; Fernando et al. 2007; Selin et al. 2010
Pseudomonas fluorescens	Bin et al. 1991; Expert and Digat 1995
Pseudomonas putida	Expert and Digat 1995
Pseudomonas cepacia (syn. Burkholderia cepacia)	McLoughlin et al. 1992
Pseudomonas brassicacearum	Savchuk and Fernando 2004; Berry et al. 2010; Ortet et al. 2011; Loewen et al. 2014; Berry et al. 2014
Serratia plymuthica	Thaning et al. 2001; Kamensky et al. 2003

Production of antimicrobial metabolites by *Pseudomonas* spp. is governed by a complex network involving multiple regulatory elements (Berry et al. 2014).

Bacillus strains were often used as biological control agents against Sclerotinia diseases (Table 2). It was observed that *Bacillus cereus* and *B. subtilis* reduced hyphal growth of the pathogen and minimized sclerotinia stem rot disease incidence in sunflower (Zazzerini 1987). Hu et al. (2014) demonstrated that *B. subtilis* BY-2 suppressed a disease of oilseed rape caused by *S. sclerotiorum* when applied as seed coating or as a spray at flowering. The mean disease incidence in the treatment with *B. subtilis* BY-2 was 8.9-11.8%, while it was 18.1-22.9% in the control. Kamal et al. (2015) showed that two applications of *B. cereus* SC-1 at 7-day intervals significantly reduced the incidence of sclerotinia stem rot of canola (6.5-9.3%), compared with the control (20.0-29.8%).

Bacillus spp. produce a wide range of biological active compounds that suppress development of many plant pathogens (Zhao et al. 2012). However, a recent investigation demonstrated that the amount of antifungal or antibacterial compounds released by this bacteria in the rhizosphere is relatively low, raising doubts that a direct suppression of plant pathogens plays a major role (Chowdhury et al. 2015). More likely, it seems that the main mechanism responsible for biocontrol activity is the induced systemic resistance (ISR) triggered by compounds produced by *Bacillus* spp. (Kloepper et al. 2004).

Another bacterial species *Serratia plymuthica IC14* that showed antifungal activity towards *S. sclerotiorum* was reported by Kamensky et al. (2003). This bacterium protected cucumber against *S. sclerotiorum* white mold disease under greenhouse conditions. *S. plymuthica* produces antibiotic pyrrolnitrin, siderophores and proteolytic as well as chitynolytic enzymes. Mutants of *S. plymuthica* deficient or with a higher production of chitynolytic enzymes had a similar effect towards the suppression of Sclerotinia foliar disease as the parental strain, suggesting that the chitynolytic enzymes are not essential for the biocontrol of *S. sclerotiorum* by *S. plymutica*. However, Thaning et al. (2001) demonstrated that *S. plymuthica* suppresses apothecial formation of *S. sclerotiorum*.

Organic amendments

It is known that organic materials added to the soil improve soil properties, plant health and yield. These substances are sources of nutrients for soil microorganisms and cause quantitative and qualitative changes in the communities of bacteria and fungi (Emmerling et al. 2002). Suppressive effects of the organic amendments against soil-borne fungal diseases have often been attributed to enhanced microbial activity (Mazzola 2004; Borneman and Becker 2007; Bonanomi et al. 2007). One of the methods for elimination of fungal propagules from infested soil is the application of organic material containing biological active compounds (Huang and Huang 1993; Gamliel et al. 2000; Smolinska 2000; Huang et al. 2002, 2005; Smolinska et al. 2016). Volatile and non-volatile compounds formed during decomposition of these material in the soil may exhibit toxic effect towards many microorganisms. The quantity and quality of compounds formed during decomposition of organic materials depend on several physical, chemical and biological processes taking place in the soil. Huang et al. (2002) tested 87 organic residues to assess their potential for controlling carpogenic germination of sclerotia *S. sclerotiorum*. Among them, 46 effectively inhibited the development of the fungus when the materials were applied to the soil at a dose of 3% w/w. However, only three kinds of residues were effective at 0.5% w/w. The most effective in preventing ascospore production were materials with elevated levels of nitrogen, e.g. fish meal. The authors suggested that the loss of viability of sclerotia in the soil was connected with the production of ammonia and ammonia-related compounds.

Addition of organic materials to soils infested by a pathogen may have positive effects when they stimulate the antagonistic microorganisms or negative effects when they increase the population of pathogens (Bonanomi et al. 2007). Ferraz et al. (1999) demonstrated that soils rich in organic matter stimulated carpogenic germination of *S. sclerotiorum* sclerotia. One of the most promising methods of *Sclerotinia* elimination from infested field soil and avoiding the danger of pathogen multiplication is the application of organic materials together with antagonistic microorganisms. The study by Huang et al. (2002) reveals that amendment of soil with organic residues infested with *C. minitans* or *Trichoderma virens* decreased carpogenic germination of sclerotia. Furthermore, Smolinska et al. (2016) reported that the application of selected *Trichoderma* species on organic carriers prepared from agro-industrial wastes (wheat straw, apple and strawberry pomaces, potato pulp, dry onion rind, rapeseed meal), allowed the complete eradication of *S. sclerotiorum* sclerotia. Organic compounds provide nutrients for mycoparasitic fungi which allows the maintenance of their population in the soil for a long time at a high level. On the other hand, overgrowing of plant residues with *Trichoderma* prevents pathogen reproduction on these materials. Bonanomi et al. (2007), after analysis of about 2500 experiments, concluded, that in the case of *S. sclerotiorum*, the population of the pathogen increased in over 50% of the cases after addition of organic amendments. In conducive conditions for Sclerotinia, the addition of plant residues to the soil infested with sclerotia significantly decreased the yield of lettuce plants (Smolinska et al. 2016).

Conclusion

The growing cultivation of plants particularly sensitive to Sclerotinia diseases (canola, carrot, sunflower, bean, lettuce

and other) causes the accumulation of *S. sclerotiorum* sclerotia in field soils and increases crop losses all over the world. The effectiveness of biological control methods is rarely sufficient to completely reduce the population of the pathogen. Disease restriction is possible only if the concentration of the pathogen is not too high. The most promising method seems to be the application of antagonistic fungi with strong parasitic properties, e.g. *C. minitans* or fungi of the genus *Trichoderma* on organic carriers which extend their persistence in the soil.

The consensus is that the application of biological methods seems to be safer for the environment than the use of synthetic pesticides. However, most likely the effective control of this pathogen will require for a long time the application of combined methods: chemical and biological protection, crop rotation and the use of resistant cultivars.

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